Part 12: Pediatric Advanced Life Support

Circulation published online Nov 28, 2005;
DOI: 10.1161/CIRCULATIONAHA.105.166573
Circulation is published by the American Heart Association. 7272 Greenville Avenue, Dallas, TX 72514
 Print ISSN: 0009-7322. Online ISSN: 1524-4539

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In contrast to adults, sudden cardiac arrest in children is uncommon, and cardiac arrest does not usually result from a primary cardiac cause.[1] More often it is the terminal event of progressive respiratory failure or shock, also called an asphyxial arrest.

Respiratory Failure

Respiratory failure is characterized by inadequate ventilation or oxygenation. Anticipate respiratory failure and possible respiratory arrest if you see any of the following:

- An increased respiratory rate, particularly with signs of distress (eg, increased effort, nasal flaring, retractions, or grunting)
- An inadequate respiratory rate, effort, or chest excursion (eg, diminished breath sounds, gasping, and cyanosis), especially if mental status is depressed

Shock

Shock results from inadequate blood flow and oxygen delivery to meet tissue metabolic demands. Shock progresses over a continuum of severity, from a compensated to a decompensated state. Attempts to compensate include tachycardia and increased systemic vascular resistance (vasoconstriction) in an effort to maintain cardiac output and blood pressure. Although decompensation can occur rapidly, it is usually preceded by a period of inadequate end-organ perfusion.

Signs of compensated shock include

- Tachycardia
- Cool extremities
- Prolonged capillary refill (despite warm ambient temperature)
- Weak peripheral pulses compared with central pulses
- Normal blood pressure

As compensatory mechanisms fail, signs of inadequate end-organ perfusion develop. In addition to the above, these signs include

- Depressed mental status
- Decreased urine output
- Metabolic acidosis
- Tachypnea
- Weak central pulses

Signs of decompensated shock include the signs listed above plus hypotension. In the absence of blood pressure measurement, decompensated shock is indicated by the nondetectable distal pulses with weak central pulses in an infant or child with other signs and symptoms consistent with inadequate tissue oxygen delivery.

The most common cause of shock is hypovolemia, one form of which is hemorrhagic shock. Distributive and cardiogenic shock are seen less often.

Learn to integrate the signs of shock because no single sign confirms the diagnosis. For example:

- Capillary refill time alone is not a good indicator of circulatory volume, but a capillary refill time of >2 seconds is a useful indicator of moderate dehydration when combined with a decreased urine output, absent tears, dry mucous membranes, and a generally ill appearance (Class IIb; LOE 3[2]). It is influenced by ambient temperature,[3] lighting,[4] site, and age.
- Tachycardia also results from other causes (eg, pain, anxiety, fever).
- Pulses may be bounding in anaphylactic, neurogenic, and septic shock.

In compensated shock, blood pressure remains normal; it is low in decompensated shock. Hypotension is a *systolic* blood pressure less than the 5th percentile of normal for age, namely:

- <60 mm Hg in term neonates (0 to 28 days)
- <70 mm Hg in infants (1 month to 12 months)
- <70 mm Hg + (2 × age in years) in children 1 to 10 years
- <90 mm Hg in children ≥10 years of age

Airway

Oropharyngeal and Nasopharyngeal Airways

Oropharyngeal and nasopharyngeal airways are adjuncts for maintaining an open airway. Oropharyngeal airways are used in unconscious victims (ie, with no gag reflex). Select the correct size: an oropharyngeal airway that is too small will not keep the tongue from obstructing the pharynx; one that is too large may obstruct the airway.

Nasopharyngeal airways will be better tolerated than oral airways by patients who are not deeply unconscious. Small nasopharyngeal tubes (for infants) may be easily obstructed by secretions.

Laryngeal Mask Airway

There is insufficient evidence to recommend for or against the routine use of a laryngeal mask airway (LMA) during cardiac arrest (Class Indeterminate). When endotracheal intubation is not possible, the LMA is an acceptable adjunct for experienced providers (Class IIb; LOE 7),[5] but it is associated with a higher incidence of complications in young children.[6]

Breathing: Oxygenation and Assisted Ventilation

For information about the role of ventilation during CPR, see Part 11: "Pediatric Basic Life Support."

(*Circulation.* 2005;112:IV-167-IV-187.)

This special supplement to *Circulation* is freely available at http://www.circulationaha.org

DOI: 10.1161/CIRCULATIONAHA.105.166573

Oxygen

There are no studies comparing various concentrations of oxygen during resuscitation beyond the perinatal period. Use 100% oxygen during resuscitation (Class Indeterminate). Monitor the patient's oxygen level. When the patient is stable, wean the supplementary oxygen if the oxygen saturation is maintained.

Pulse Oximetry

If the patient has a perfusing rhythm, monitor oxygen saturation continuously with a pulse oximeter because clinical recognition of hypoxemia is not reliable.[7] Pulse oximetry, however, may be unreliable in a patient with poor peripheral perfusion.

Bag-Mask Ventilation

Bag-mask ventilation can be as effective as ventilation through an endotracheal tube for short periods and may be safer.[8–11] In the prehospital setting ventilate and oxygenate infants and children with a bag-mask device, especially if transport time is short (Class IIa; LOE 1[8]; 3[10]; 4[9,11]). Bag-mask ventilation requires training and periodic retraining on how to select a correct mask size, open the airway, make a tight seal between mask and face, ventilate, and assess effectiveness of ventilation (see Part 11: "Pediatric Basic Life Support").

Precautions

Victims of cardiac arrest are frequently overventilated during resuscitation.[12–14] Excessive ventilation increases intrathoracic pressure and impedes venous return, reducing cardiac output, cerebral blood flow, and coronary perfusion.[13] Excessive ventilation also causes air trapping and barotrauma in patients with small-airway obstruction and increases the risk of stomach inflation, regurgitation, and aspiration.

Minute ventilation is determined by the tidal volume and ventilation rate. Use only the force and tidal volume needed to make the chest rise visibly. During CPR for the patient with no advanced airway (eg, endotracheal tube, esophageal-tracheal combitube [Combitube], LMA) in place, ventilation rate is determined by the compression-ventilation ratio. Pause after 30 compressions (1 rescuer) or after 15 compressions (2 rescuers) to give 2 ventilations with mouth-to-mouth, mouth-to-mask, or bag-mask techniques. Give each breath over 1 second.

If an advanced airway is in place during CPR (eg, endotracheal tube, Combitube, LMA), ventilate at a rate of 8 to 10 times per minute without pausing chest compressions. In the victim with a perfusing rhythm but absent or inadequate respiratory effort, give 12 to 20 breaths per minute. One way to achieve this rate with a ventilating bag is to use the mnemonic "squeeze-release-release" at a normal speaking rate.[8,15]

Two-Person Bag-Mask Ventilation

A 2-person technique may be more effective than ventilation by a single rescuer if the patient has significant airway obstruction, poor lung compliance, or difficulty in creating a tight mask-to-face seal.[16,17] One rescuer uses both hands to maintain an open airway with a jaw thrust and a tight mask-to-face seal while the other compresses the ventilation bag. Both rescuers should observe the victim's chest to ensure chest rise.

Gastric Inflation

Gastric inflation may interfere with effective ventilation[18] and cause regurgitation. You can minimize gastric inflation by doing the following:

- Avoid excessive peak inspiratory pressures (eg, by ventilating slowly and watching chest rise).[8] To avoid use of excessive volume, deliver only the volume needed to produce visible chest rise.
- Apply cricoid pressure. You should do so only in an unresponsive victim. This technique may require an additional (third) rescuer if the cricoid pressure cannot be applied by the rescuer who is securing the bag to the face.[19–21] Avoid excessive pressure so as not to obstruct the trachea.[22]
- If you intubate the patient, pass a nasogastric or orogastric tube *after* you intubate because a gastric tube interferes with the gastroesophageal sphincter, allowing possible regurgitation.

Ventilation Through an Endotracheal Tube

Endotracheal intubation in infants and children requires special training because the pediatric airway anatomy differs from adult airway anatomy. Success and a low complication rate are related to the length of training, supervised experience in the operating room and in the field,[23,24] adequate ongoing experience,[25] and the use of rapid sequence intubation (RSI).[23,26,27]

Rapid Sequence Intubation

To facilitate emergency intubation and reduce the incidence of complications, skilled, experienced providers may use sedatives, neuromuscular blocking agents, and other medications to rapidly sedate and paralyze the victim.[28] Use RSI only if you are trained and have experience using these medications and are proficient in the evaluation and management of the pediatric airway. If you use RSI you must have a secondary plan to manage the airway in the event that you cannot achieve intubation.

Cuffed Versus Uncuffed Tubes

In the in-hospital setting a cuffed endotracheal tube is as safe as an uncuffed tube for infants beyond the newborn period and in children.[29–31] In certain circumstances (eg, poor lung compliance, high airway resistance, or a large glottic air leak) a cuffed tube may be preferable provided that attention is paid to endotracheal tube size, position, and cuff inflation pressure (Class IIa; LOE 2[30]; 3[29,31]). Keep cuff inflation pressure <20 cm H_2O.[32]

Endotracheal Tube Size

The internal diameter of the appropriate endotracheal tube for a child will roughly equal the size of that child's little finger, but this estimation may be difficult and unreliable.[33,34] Several formulas such as the ones below allow estimation of proper endotracheal tube size (ID, internal diameter) for children 1 to 10 years of age, based on the child's age:

Uncuffed endotracheal tube size (mm ID)
=(age in years/4) + 4

In general, during preparation for intubation using the above formula, providers should have the estimated tube size available, as well as uncuffed endotracheal tubes that have internal diameters that are 0.5 mm smaller and 0.5 mm larger than the size estimated ready at the bedside for use.

The formula for estimation of a cuffed endotracheal tube size is as follows[30]:

Cuffed endotracheal tube size (mm ID)
= (age in years/4) + 3

Endotracheal tube size, however, is more reliably based on a child's *body length.* Length-based resuscitation tapes are helpful for children up to approximately 35 kg.[35]

Verification of Endotracheal Tube Placement

There is a high risk that an endotracheal tube will be misplaced (ie, placed in the esophagus or in the pharynx above the vocal chords), displaced, or become obstructed,[8,36] especially when the patient is moved.[37] No single confirmation technique, including clinical signs[38] or the presence of water vapor in the tube,[39] is completely reliable, so providers must use both clinical assessment and confirmatory devices to verify proper tube placement immediately after intubation, during transport, and when the patient is moved (ie, from gurney to bed).

Immediately after intubation and again after securing the tube, confirm correct tube position with the following techniques while you provide positive-pressure ventilation with a bag:

- Look for bilateral chest movement and listen for equal breath sounds over both lung fields, especially over the axillae.
- Listen for gastric insufflation sounds over the stomach (they should not be present if the tube is in the trachea).[38]
- Use a device to evaluate placement. Check for exhaled CO_2 (see below) if there is a perfusing rhythm. If the child has a perfusing rhythm and is >20 kg, you may use an esophageal detector device to check for evidence of esophageal placement (see below).
- Check oxygen saturation with a pulse oximeter. Following hyperoxygenation, the oxyhemoglobin saturation detected by pulse oximetry may not demonstrate a fall indicative of incorrect endotracheal tube position (ie, tube misplacement or displacement) for as long as 3 minutes.[40,41]
- If you are still uncertain, perform direct laryngoscopy and look to see if the tube goes between the cords.
- In hospital settings perform a chest x-ray to verify that the tube is not in the right main bronchus and to identify a high tube position at risk of easy displacement.

After intubation secure the tube. There is insufficient evidence to recommend any one method (Class Indeterminate). After you secure the tube, maintain the patient's head in a neutral position; neck flexion pushes the tube farther into the airway, and extension pulls the tube out of the airway.[42,43]

If an intubated patient's condition deteriorates, consider the following possibilities (**DOPE**):

- **D**isplacement of the tube from the trachea
- **O**bstruction of the tube
- **P**neumothorax
- **E**quipment failure

Exhaled or End-Tidal CO_2 Monitoring

In infants and children with a perfusing rhythm, use a colorimetric detector or capnography to detect exhaled CO_2 to confirm endotracheal tube position in the prehospital and in-hospital settings (Class IIa; LOE 5[44]) and during intrahospital and interhospital transport (Class IIb; LOE 5[45]). A color change or the presence of a capnography waveform confirms tube position in the trachea but does not rule out right main bronchus intubation. During cardiac arrest, if exhaled CO_2 is not detected, confirm tube position with direct laryngoscopy (Class IIa; LOE 5[46–49]; 6[50]) because the absence of CO_2 may be a reflection of low pulmonary blood flow.

You may also detect a low end-tidal CO_2 in the following circumstances:

- If the detector is contaminated with gastric contents or acidic drugs (eg, endotracheally administered epinephrine), you may see a constant color rather than breath-to-breath color change.
- An intravenous (IV) bolus of epinephrine may transiently reduce pulmonary blood flow and exhaled CO_2 below the limits of detection.[51]
- Severe airway obstruction (eg, status asthmaticus) and pulmonary edema may impair CO_2 elimination.[49,52–54]

Esophageal Detector Devices

The self-inflating bulb (esophageal detector device) may be considered to confirm endotracheal tube placement in children weighing >20 kg with a perfusing rhythm (Class IIb; LOE 2[55,56]). There is insufficient data to make a recommendation for or against its use in children during cardiac arrest (Class Indeterminate).

Transtracheal Catheter Ventilation

Transtracheal catheter ventilation may be considered for support of oxygenation in the patient with severe airway obstruction if you cannot provide oxygen or ventilation any other way. Try transtracheal ventilation only if you are properly trained and have appropriate equipment.[57]

Suction Devices

A suction device with an adjustable suction regulator should be available. Use a maximum suction force of 80 to 120 mm Hg for suctioning the airway via an endotracheal tube.[58] You will need higher suction pressures and large-bore noncollapsible suction tubing as well as semirigid pharyngeal tips to suction the mouth and pharynx.

Circulation

Advanced cardiovascular life support techniques are useless without effective circulation, which is supported by good chest compressions during cardiac arrest. Good chest compressions require an adequate compression rate (100 com-

pressions per minute), an adequate compression depth (about one third to one half of the anterior-posterior diameter), full recoil of the chest after each compression, and minimal interruptions in compressions. Unfortunately, good compressions are not always performed for many reasons,[14] including rescuer fatigue and long or frequent interruptions to secure the airway, check the heart rhythm, and move the patient.

Backboard

A firm surface that extends from the shoulders to the waist and across the full width of the bed provides optimal support for effective chest compressions. In ambulances and mobile life support units, use a spine board.[59,60]

CPR Techniques and Adjuncts

There is insufficient data to make a recommendation for or against the use of mechanical devices to compress the sternum, active compression-decompression CPR, interposed abdominal compression CPR, pneumatic antishock garment during resuscitation from cardiac arrest, and open-chest direct heart compression (Class Indeterminate). For further information see Part 6: "CPR Techniques and Devices."

Extracorporeal Membrane Oxygenation

Consider extracorporeal CPR for in-hospital cardiac arrest refractory to initial resuscitation attempts if the condition leading to cardiac arrest is reversible or amenable to heart transplantation, if excellent conventional CPR has been performed after no more than several minutes of no-flow cardiac arrest (arrest time without CPR), and if the institution is able to rapidly perform extracorporeal membrane oxygenation (Class IIb; LOE 5[61,62]). Long-term survival is possible even after >50 minutes of CPR in selected patients.[61,62]

Cardiovascular Monitoring

Attach electrocardiographic (ECG) monitoring leads or defibrillator pads as soon as possible and monitor blood pressure. If the patient has an indwelling arterial catheter, use the waveform to guide your technique in compressing the chest. A minor adjustment of your hand position or depth of compression can significantly improve the waveform.

Vascular Access

Vascular access is essential for administering medications and drawing blood samples. Venous access can be challenging in infants and children during an emergency, whereas intraosseous (IO) access can be easily achieved. Limit the time you attempt venous access,[63] and if you cannot achieve reliable access quickly, establish IO access. In cardiac arrest immediate IO access is recommended if no other IV access is already in place.

Intraosseous Access

IO access is a rapid, safe, and effective route for the administration of medications and fluids,[64,65] and it may be used for obtaining an initial blood sample during resuscitation (Class IIa; LOE 3[65,66]). You can safely administer epinephrine, adenosine, fluids, blood products,[64,66] and catecholamines.[67] Onset of action and drug levels achieved are comparable to venous administration.[68] You can also obtain blood specimens for type and crossmatch and for chemical and blood gas analysis even during cardiac arrest,[69] but acid-base analysis is inaccurate after sodium bicarbonate administration via the IO cannula.[70] Use manual pressure or an infusion pump to administer viscous drugs or rapid fluid boluses,[71,72] and follow each medication with a saline flush to promote entry into the central circulation.

Venous Access

A central intravenous line (IV) provides more secure long-term access, but central drug administration does not achieve higher drug levels or a substantially more rapid response than peripheral administration.[73]

Endotracheal Drug Administration

Any vascular access, IO or IV, is preferable, but if you cannot establish vascular access, you can give lipid-soluble drugs such as lidocaine, epinephrine, atropine, and naloxone ("LEAN")[74,75] via the endotracheal tube,[76] although optimal endotracheal doses are unknown (Table 1). Flush with a minimum of 5 mL normal saline followed by 5 assisted manual ventilations.[77] If CPR is in progress, stop chest compressions briefly during administration of medications. Although naloxone and vasopressin may be given by the endotracheal route, there are no human studies to support a specific dose. Non–lipid-soluble drugs (eg, sodium bicarbonate and calcium) may injure the airway and should not be administered via the endotracheal route.

Administration of resuscitation drugs into the trachea results in lower blood concentrations than the same dose given intravascularly. Furthermore, recent animal studies suggest that the lower epinephrine concentrations achieved when the drug is delivered by the endotracheal route may produce transient β-adrenergic effects. These effects can be detrimental, causing hypotension, lower coronary artery perfusion pressure and flow, and reduced potential for return of spontaneous circulation. Thus, although endotracheal administration of some resuscitation drugs is possible, IV or IO drug administration is preferred because it will provide a more predictable drug delivery and pharmacologic effect.

Emergency Fluids and Medications

Estimating Weight

In the out-of-hospital setting a child's weight is often unknown, and even experienced personnel may not be able to estimate it accurately.[78] Tapes with precalculated doses printed at various patient lengths are helpful and have been clinically validated.[35,78,79] Hospitalized patients should have weights and precalculated emergency drug doses recorded and readily available.

Fluids

Use an isotonic crystalloid solution (eg, lactated Ringer's solution or normal saline)[80,81] to treat shock; there is no benefit in using colloid (eg, albumin) during initial resuscitation.[82] Use bolus therapy with a glucose-containing solution to only treat documented hypoglycemia (Class IIb; LOE 2[83]; 6[84]). There is insufficient data to make a recommendation for

TABLE 1. Medications for Pediatric Resuscitation and Arrhythmias

Medication	Dose	Remarks
Adenosine	0.1 mg/kg (maximum 6 mg) Repeat: 0.2 mg/kg (maximum 12 mg)	Monitor ECG Rapid IV/IO bolus
Amiodarone	5 mg/kg IV/IO; repeat up to 15 mg/kg Maximum: 300 mg	Monitor ECG and blood pressure Adjust administration rate to urgency (give more slowly when perfusing rhythm present) Use caution when administering with other drugs that prolong QT (consider expert consultation)
Atropine	0.02 mg/kg IV/IO 0.03 mg/kg ET* Repeat once if needed Minimum dose: 0.1 mg Maximum single dose: Child 0.5 mg Adolescent 1 mg	Higher doses may be used with organophosphate poisoning
Calcium chloride (10%)	20 mg/kg IV/IO (0.2 mL/kg)	Slowly Adult dose: 5–10 mL
Epinephrine	0.01 mg/kg (0.1 mL/kg 1:10 000) IV/IO 0.1 mg/kg (0.1 mL/kg 1:1000) ET* Maximum dose: 1 mg IV/IO; 10 mg ET	May repeat q 3–5 min
Glucose	0.5–1 g/kg IV/IO	$D_{10}W$: 5–10 mL/kg $D_{25}W$: 2–4 mL/kg $D_{50}W$: 1–2 mL/kg
Lidocaine	Bolus: 1 mg/kg IV/IO Maximum dose: 100 mg Infusion: 20–50 μg/kg per minute ET*: 2–3 mg	
Magnesium sulfate	25–50 mg/kg IV/IO over 10–20 min; faster in torsades Maximum dose: 2g	
Naloxone	$<$5 y or $\leq$20 kg: 0.1 mg/kg IV/IO/ET* $\geq$5 y or $>$20 kg: 2 mg IV/IO/ET*	Use lower doses to reverse respiratory depression associated with therapeutic opioid use (1–15 μg/kg)
Procainamide	15 mg/kg IV/IO over 30–60 min Adult dose: 20 mg/min IV infusion up to total maximum dose 17 mg/kg	Monitor ECG and blood pressure Use caution when administering with other drugs that prolong QT (consider expert consultation)
Sodium bicarbonate	1 mEq/kg per dose IV/IO slowly	After adequate ventilation

IV indicates intravenous; IO, intraosseous; and ET, via endotracheal tube.
*Flush with 5 mL of normal saline and follow with 5 ventilations.

or against hypertonic saline for shock associated with head injuries or hypovolemia (Class Indeterminate).[85,86]

Medications (See Table 1)

Adenosine

Adenosine causes a temporary atrioventricular (AV) nodal conduction block and interrupts reentry circuits that involve the AV node. It has a wide safety margin because of its short half-life.

A higher dose may be required for peripheral administration than central venous administration.[87,88] Based on experimental data[89] and a case report,[90] adenosine may also be given by IO route. Administer adenosine and follow with a rapid saline flush to promote flow toward the central circulation.

Amiodarone

Amiodarone slows AV conduction, prolongs the AV refractory period and QT interval, and slows ventricular conduction (widens the QRS).

Precautions

Monitor blood pressure and administer as slowly as the patient's clinical condition allows; it should be administered slowly to a patient with a pulse but may be given rapidly to a patient with cardiac arrest or ventricular fibrillation (VF). Amiodarone causes hypotension through its vasodilatory property. The severity of the hypotension is related to the infusion rate and is less common with the aqueous form of amiodarone.[91]

Monitor the ECG because complications may include bradycardia, heart block, and torsades de pointes ventricular tachycardia (VT). Use extreme caution when administering with another drug causing QT prolongation, such as procainamide. Consider obtaining expert consultation. Adverse effects may be long lasting because the half-life is up to 40 days.[92]

Atropine

Atropine sulfate is a parasympatholytic drug that accelerates sinus or atrial pacemakers and increases AV conduction.

Precautions
Small doses of atropine (<0.1 mg) may produce paradoxical bradycardia.[93] Larger than recommended doses may be required in special circumstances (eg, organophosphate poisoning[94] or exposure to nerve gas agents).

Calcium
Routine administration of calcium does not improve outcome of cardiac arrest.[95] In critically ill children, calcium chloride may provide greater bioavailability than calcium gluconate.[96] Preferably administer calcium chloride via a central venous catheter because of the risk of sclerosis or infiltration with a peripheral venous line.

Epinephrine
The α-adrenergic-mediated vasoconstriction of epinephrine increases aortic diastolic pressure and thus coronary perfusion pressure, a critical determinant of successful resuscitation.[97,98]

Precautions
Administer all catecholamines through a secure line, preferably into the central circulation; local ischemia, tissue injury, and ulceration may result from tissue infiltration.

Do not mix catecholamines with sodium bicarbonate; alkaline solutions inactivate them.

In patients with a perfusing rhythm, epinephrine causes tachycardia and may cause ventricular ectopy, tachyarrhythmias, hypertension, and vasoconstriction.[99]

Glucose
Infants have high glucose requirements and low glycogen stores and develop hypoglycemia when energy requirements rise.[100] Check blood glucose concentrations during and after arrest and treat hypoglycemia promptly (Class IIb; LOE 1[101]; 7 [most extrapolated from neonates and adult ICU studies]).

Lidocaine
Lidocaine decreases automaticity and suppresses ventricular arrhythmias[102] but is not as effective as amiodarone for improving intermediate outcomes (ie, return of spontaneous circulation or survival to hospital admission) among adult patients with VF refractory to a shock and epinephrine.[103] Neither lidocaine nor amiodarone has been shown to improve survival to hospital discharge among patients with VF cardiac arrest.

Precautions
Lidocaine toxicity includes myocardial and circulatory depression, drowsiness, disorientation, muscle twitching, and seizures, especially in patients with poor cardiac output and hepatic or renal failure.[104,105]

Magnesium
There is insufficient evidence to recommend for or against the routine administration of magnesium during cardiac arrest (Class Indeterminate).[106–108] Magnesium is indicated for the treatment of documented hypomagnesemia or for torsades de pointes (polymorphic VT associated with long QT interval). Magnesium produces vasodilation and may cause hypotension if administered rapidly.

Procainamide
Procainamide prolongs the refractory period of the atria and ventricles and depresses conduction velocity.

Precautions
There is little clinical data on using procainamide in infants and children.[109,110] Infuse procainamide very slowly while you monitor for hypotension, prolongation of the QT interval, and heart block. Stop the infusion if the QRS widens to >50% of baseline or if hypotension develops. Use extreme caution when administering with another drug causing QT prolongation, such as amiodarone. Consider obtaining expert consultation.

Sodium Bicarbonate
The routine administration of sodium bicarbonate has not been shown to improve outcome of resuscitation (Class Indeterminate). After you have provided effective ventilation and chest compressions and administered epinephrine, you may consider sodium bicarbonate for prolonged cardiac arrest (Class IIb; LOE 6). Sodium bicarbonate administration may be used for treatment of some toxidromes (see "Toxicologic Emergencies," below) or special resuscitation situations.

During cardiac arrest or severe shock, arterial blood gas analysis may not accurately reflect tissue and venous acidosis.[111,112]

Precautions
Excessive sodium bicarbonate may impair tissue oxygen delivery[113]; cause hypokalemia, hypocalcemia, hypernatremia, and hyperosmolality[114,115]; decrease the VF threshold[116]; and impair cardiac function.

Vasopressin
There is limited experience with the use of vasopressin in pediatric patients,[117] and the results of its use in the treatment of adults with VF cardiac arrest have been inconsistent.[118–121] There is insufficient evidence to make a recommendation for or against the routine use of vasopressin during cardiac arrest (Class Indeterminate; LOE 5[117]; 6[121], 7[118–120] [extrapolated from adult literature]).

Pulseless Arrest

In the text below, box numbers identify the corresponding box in the algorithm (Figure 1.)

If a victim becomes unresponsive (Box 1), start CPR immediately (with supplementary oxygen if available) and send for a defibrillator (manual or automated external defibrillator [AED]). Asystole and bradycardia with a wide QRS complex are most common in asphyxial cardiac arrest.[1,23] VF and pulseless electrical activity (PEA) are less common[122] and more likely to be observed in children with sudden arrest. If you are using an ECG monitor, determine the rhythm (Box 2); if you are using an AED, the device will tell you whether the rhythm is "shockable" (ie, VF or rapid VT), but it may not display the rhythm.

"Shockable Rhythm": VF/Pulseless VT (Box 3)

VF occurs in 5% to 15% of all pediatric victims of out-of-hospital cardiac arrest[123–125] and is reported in up to 20% of

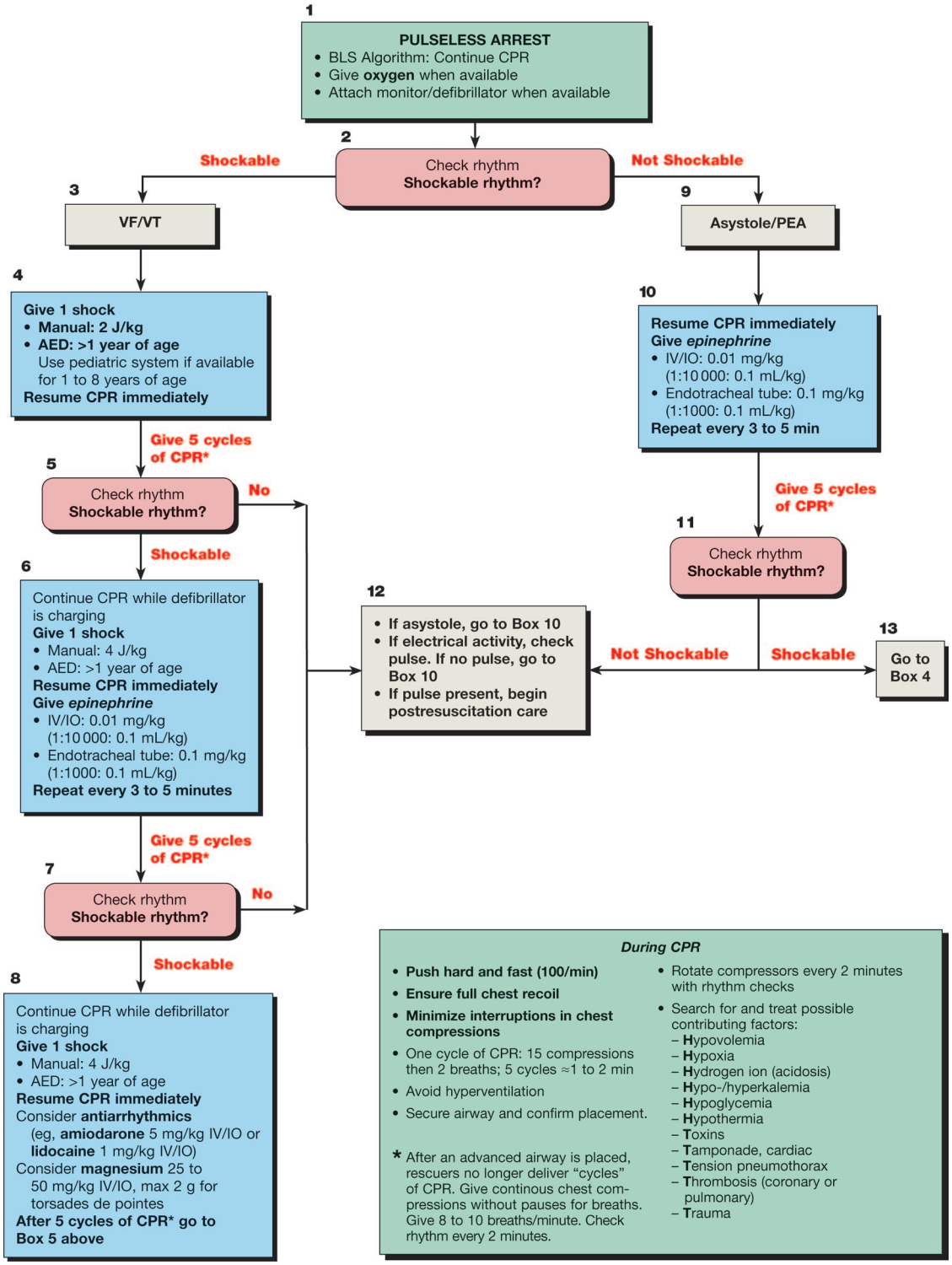

Figure 1. PALS Pulseless Arrest Algorithm.

pediatric in-hospital arrests at some point during the resuscitation. The incidence increases with age.[123,125] Defibrillation is the definitive treatment for VF (Class I) with an overall survival rate of 17% to 20%,[125–127] but in adults the probability of survival declines by 7% to 10% for each minute of arrest without CPR and defibrillation.[128] The decline in survival is more gradual when early CPR is provided.

Defibrillators

Defibrillators are either manual or automated (AED), with monophasic or biphasic waveforms. For further information see Part 5: "Electrical Therapies: Automated External Defibrillators, Defibrillation, Cardioversion, and Pacing."

Institutions that care for children at risk for arrhythmias and cardiac arrest (eg, hospitals, emergency departments) ideally should have defibrillators available that are capable of

energy adjustment that is appropriate for children. Many AED parameters are set automatically. When using a manual defibrillator, several elements should be considered, and they are highlighted below.

Paddle Size
Use the largest paddles or self-adhering electrodes[129–131] that will fit on the chest wall without touching (leave about 3 cm between the paddles). The best paddle size is

- Adult paddles (8 to 10 cm) for children >10 kg (more than approximately 1 year of age)
- Infant paddles for infants weighing <10 kg

Interface
The electrode–chest wall interface can be gel pads, electrode cream, paste, or self-adhesive monitoring-defibrillation pads. Do not use saline-soaked pads, ultrasound gel, bare paddles, or alcohol pads.

Paddle Position
Apply firm pressure on the paddles (manual) placed over the right side of the upper chest and the apex of the heart (to the left of the nipple over the left lower ribs). Alternatively place one electrode on the front of the chest just to the left of the sternum and the other over the upper back below the scapula.[132]

Energy Dose
The lowest energy dose for effective defibrillation and the upper limit for safe defibrillation in infants and children are not known. Energy doses >4 J/kg (up to 9 J/kg) have effectively defibrillated children[133–135] and pediatric animal models[136] with negligible adverse effects. Based on data from adult studies[137,138] and pediatric animal models,[139–141] biphasic shocks appear to be at least as effective as monophasic shocks and less harmful. With a manual defibrillator (monophasic or biphasic), use a dose of 2 J/kg for the first attempt (Class IIa; LOE 5[142]; 6[136]) and 4 J/kg for subsequent attempts (Class Indeterminate).

AEDs
Many AEDs can accurately detect VF in children of all ages[143–145] and differentiate shockable from nonshockable rhythms with a high degree of sensitivity and specificity.[143,144] Since publication of the *ECC Guidelines 2000*, data has shown that AEDs can be safely and effectively used in children 1 to 8 years of age.[143–146] There is insufficient data to make a recommendation for or against using an AED in infants <1 year of age (Class Indeterminate).[146] When using an AED for children about 1 to 8 years old, use a pediatric attenuator system, which decreases the delivered energy to a dose suitable for children (Class IIb; LOE 5[136]; 6[139,141]). If an AED with a pediatric attenuating system is not available, use a standard AED, preferably one with sensitivity and specificity for pediatric shockable rhythms. It is recommended that systems and institutions caring for children and having AED programs should use AEDs with both a high specificity to recognize pediatric shockable rhythms and a pediatric attenuating system.

Defibrillation Sequence (Boxes 4, 5, 6, 7, 8)
The following are important considerations:

- Attempt defibrillation immediately. The earlier you attempt defibrillation, the more likely the attempt will be successful.
- Provide CPR until the defibrillator is ready to deliver a shock, and resume CPR, beginning with chest compressions, immediately after shock delivery. Minimize interruptions of chest compressions. In adults with a prolonged arrest[147,148] and animal models,[134,149] defibrillation is more likely to be successful after a period of effective chest compressions. Ideally, chest compressions should be interrupted only for ventilations (until an advanced airway is in place), rhythm check, and shock delivery. Rescuers should provide chest compressions after a rhythm check (when possible) while the defibrillator is charging.
- Give 1 shock (2 J/kg) as quickly as possible and immediately resume CPR, beginning with chest compressions (Box 4). Biphasic defibrillators have a first shock success rate that exceeds 90%.[150] If 1 shock fails to eliminate VF, the incremental benefit of another shock is low, and resumption of CPR is likely to confer a greater value than another shock. CPR may provide some coronary perfusion with oxygen and substrate delivery, increasing the likelihood of defibrillation with a subsequent shock. It is important to minimize the time between any interruption in chest compressions and shock delivery and between shock delivery and resumption of postshock compressions. Check the rhythm (Box 5). Continue CPR for about 5 cycles (about 2 minutes). In in-hospital settings with continuous monitoring (eg, electrocardiographic, hemodynamic) in place, this sequence may be modified at the physician's discretion (see Part 7.2: "Management of Cardiac Arrest").
- Check the rhythm (Box 5). If a shockable rhythm persists, give 1 shock (4 J/kg), resume compressions immediately. Give a dose of epinephrine. The drug should be administered as soon as possible after the rhythm check. It is helpful if a third rescuer prepares the drug doses *before* the rhythm is checked so a drug can be administered as soon as possible after the rhythm is checked. A drug should be administered during the CPR that is performed while the defibrillator is charging or immediately after shock delivery. However, the timing of drug administration is less important than the need to minimize interruptions in chest compressions.

  Use a standard dose of epinephrine for the first and subsequent doses (Class IIa; LOE 4).[151] There is no survival benefit from routine use of high-dose epinephrine, and it may be harmful, particularly in asphyxia (Class III; LOE 2, 4).[151] High-dose epinephrine may be considered in exceptional circumstances, such as β-blocker overdose (Class IIb). Give the standard dose of epinephrine about every 3 to 5 minutes during cardiac arrest.
- After 5 cycles (approximately 2 minutes) of CPR, check the rhythm (Box 7). If the rhythm continues to be "shockable," deliver a shock (4 J/kg), resume CPR (beginning with chest compressions) immediately, and give amiodarone (Class IIb; LOE 3, 7)[103, 152–154] or lidocaine if you

do not have amiodarone (Box 8) while CPR is provided. Continue CPR for 5 cycles (about 2 minutes) before again checking the rhythm and attempting to defibrillate if needed with 4 J/kg (you now have returned to Box 6).

- Once an advanced airway is in place, 2 rescuers no longer deliver cycles of CPR (ie, compressions interrupted by pauses for ventilation). Instead, the compressing rescuer should give continuous chest compressions at a rate of 100 per minute without pauses for ventilation. The rescuer delivering ventilation provides 8 to 10 breaths per minute. Two or more rescuers should rotate the compressor role approximately every 2 minutes to prevent compressor fatigue and deterioration in quality and rate of chest compressions.
- If you have a monitor or an AED with a rhythm display and there is an organized rhythm at any time, check for a pulse and proceed accordingly (Box 12).
- If defibrillation is successful but VF recurs, continue CPR while you give another bolus of amiodarone before you try to defibrillate with the previously successful shock dose (see Box 8).
- Search for and treat reversible causes (see green "During CPR" box).

Torsades de Pointes

This polymorphic VT is seen in patients with a long QT interval, which may be congenital or may result from toxicity with type I_A antiarrhythmics (eg, procainamide, quinidine, and disopyramide) or type III antiarrhythmics (eg, sotalol and amiodarone), tricyclic antidepressants (see below), digitalis, or drug interactions.[155,156] These are examples of contributing factors listed in the green box in the algorithm.

Treatment

Regardless of the cause, treat torsades de pointes with a rapid (over several minutes) IV infusion of magnesium sulfate.

"Nonshockable Rhythm": Asystole/PEA (Box 9)

The most common ECG findings in infants and children in cardiac arrest are asystole and PEA. PEA is organized electrical activity—most commonly slow, wide QRS complexes—without palpable pulses. Less frequently there is a sudden impairment of cardiac output with an initially normal rhythm but without pulses and with poor perfusion. This subcategory (formerly known as electromechanical dissociation [EMD]) is more likely to be treatable. For asystole and PEA:

- Resume CPR and continue with as few interruptions in chest compressions as possible (Box 10). A second rescuer gives epinephrine while the first continues CPR. As with VF/pulseless VT, there is no survival benefit from routine high-dose epinephrine, and it may be harmful, particularly in asphyxia (Class III; LOE 2[151]; 6[99,157,158]; 7[159]). Use a standard dose for the first and subsequent doses (Class IIa; LOE 4).[151] High-dose epinephrine may be considered in exceptional circumstances such as β-blocker overdose (Class IIb).
- Search for and treat reversible causes (see the green box).

Bradycardia

Box numbers in the text below refer to the corresponding boxes in the PALS Bradycardia Algorithm (Figure 2).

The emergency treatment of bradycardia depends on its hemodynamic consequences.

- This algorithm applies to the care of the patient with bradycardia that is causing cardiorespiratory compromise (Box 1). If at any time the patient develops pulseless arrest, see the PALS Pulseless Arrest Algorithm.
- Support airway, breathing, and circulation as needed, administer oxygen, and attach a monitor/defibrillator (Box 2).
- Reassess the patient to determine if bradycardia is still causing cardiorespiratory symptoms despite support of adequate oxygenation and ventilation (Box 3).
- If pulses, perfusion, and respirations are normal, no emergency treatment is necessary. Monitor and proceed with evaluation (Box 5A).
- If heart rate is <60 beats per minute with poor perfusion despite effective ventilation with oxygen, start chest compressions (Box 6).
- Reevaluate the patient to determine if signs of hemodynamic compromise persist despite the support of adequate oxygenation and ventilation and compressions if indicated (Box 5). Verify that the support is adequate—eg, check airway and oxygen source and effectiveness of ventilation.
- Medications and pacing (Box 6)
  - —Continue to support airway, ventilation, oxygenation (and provide compressions as needed) and give epinephrine (Class IIa; LOE 7, 8). If bradycardia persists or responds only transiently, consider a continuous infusion of epinephrine or isoproterenol.
  - —If bradycardia is due to vagal stimulation, give atropine (Class I) (Box 6). Emergency transcutaneous pacing may be lifesaving if the bradycardia is due to complete heart block or sinus node dysfunction unresponsive to ventilation, oxygenation, chest compressions, and medications, especially if it is associated with congenital or acquired heart disease (Class IIb; LOE 5, 7).[160] Pacing is not useful for asystole[160,161] or bradycardia due to post-arrest hypoxic/ischemic myocardial insult or respiratory failure.

Tachycardia and Hemodynamic Instability

The box numbers in the text below correspond to the numbered boxes in the Tachycardia Algorithm (Figure 3)

If there are no palpable pulses, proceed with the PALS Pulseless Arrest Algorithm. If pulses are palpable and the patient has signs of hemodynamic compromise (poor perfusion, tachypnea, weak pulses), ensure that the airway is patent, assist ventilations if necessary, administer supplementary oxygen, and attach an ECG monitor or defibrillator (Box 1). Assess QRS duration (Box 2): determine if the QRS duration is ≤0.08 second (narrow-complex tachycardia) or >0.08 second (wide-complex tachycardia).

Narrow-Complex (≤0.08 Second) Tachycardia

Evaluation of a 12-lead ECG (Box 3) and the patient's clinical presentation and history (Boxes 4 and 5) should help

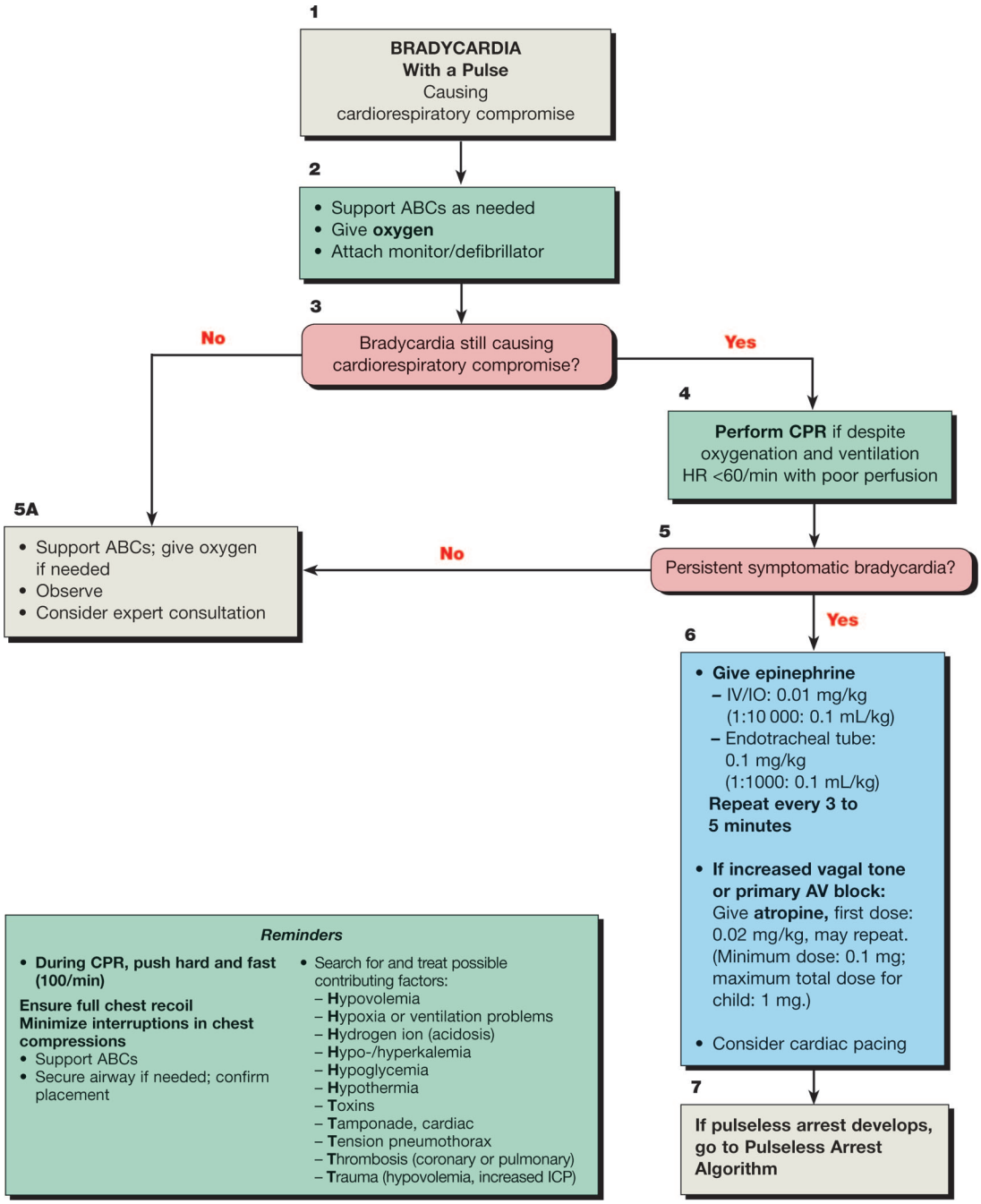

Figure 2. PALS Bradycardia Algorithm.

you differentiate probable sinus tachycardia from probable supraventricular tachycardia (SVT). If the rhythm is sinus tachycardia, search for and treat reversible causes.

Probable Supraventricular Tachycardia (Box 5)

Monitor rhythm during therapy to evaluate effect. The choice of therapy depends on the patient's degree of hemodynamic instability.

- Attempt *vagal stimulation* (Box 7) first unless the patient is very unstable and if it does not unduly delay chemical or electrical cardioversion (Class IIa; LOE 4, 5, 7, 8). In infants and young children, apply ice to the face without occluding the airway.[162,163] In older children, carotid sinus massage or Valsalva maneuvers are safe (Class IIb; LOE 5, 7).[164–166] One method of a Valsalva maneuver is to have the child blow through an obstructed straw.[165] Do not apply pressure to the eye because this can damage the retina.
- Chemical cardioversion with adenosine (Box 8) is very effective (Class IIa; LOE 2[87]; 3[88]; 7 [extrapolation from adult studies]). If IV access is readily available administer adenosine using 2 syringes connected to a T-connector or stopcock; give adenosine rapidly with one syringe and immediately flush with ≥5 mL of normal saline with the other.

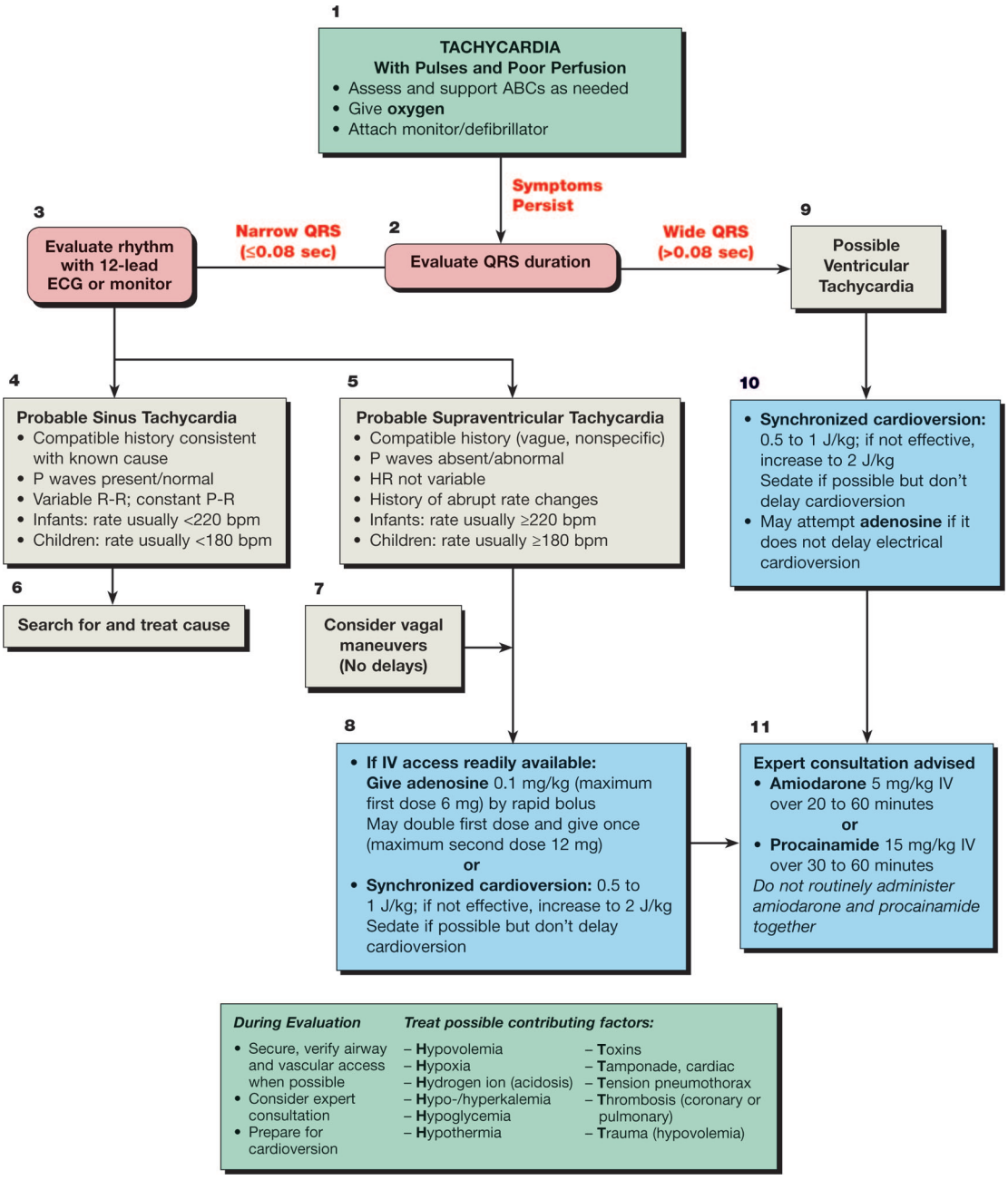

Figure 3. PALS Tachycardia Algorithm.

- If the patient is very unstable or IV access is not readily available, provide electrical (synchronized) cardioversion (Box 8). Consider sedation if possible. Start with a dose of 0.5 to 1 J/kg. If unsuccessful, repeat using a dose of 2 J/kg. If a second shock is unsuccessful or the tachycardia recurs quickly, consider antiarrhythmic therapy (amiodarone or procainamide) before a third shock.
- Consider amiodarone or procainamide (Box 11) for SVT unresponsive to vagal maneuvers and adenosine (Class IIb; 5[153, 154]; 6[167–169]; 7 [extrapolated from LOE 2 adult studies][103,152]). Use extreme caution when administering more than one drug that causes QT prolongation (eg, amiodarone and procainamide). Consider obtaining expert consultation. Give an infusion of amiodarone or procainamide slowly (over several minutes to an hour), depending on the urgency, while you monitor the ECG and blood pressure. If there is no effect and there are no signs of toxicity, give additional doses (Table 1).
- Do not use verapamil in infants because it may cause refractory hypotension and cardiac arrest (Class III; LOE 5[170,171]), and use with caution in children because it may cause hypotension and myocardial depression.[172]

Wide-Complex (>0.08 Second) Tachycardia (Box 9)

Wide-complex tachycardia with poor perfusion is probably ventricular in origin but may be supraventricular with aberrancy.[173]

- Treat with synchronized electrical cardioversion (0.5 J to 1 J/kg). If it does not delay cardioversion, try a dose of

adenosine first to determine if the rhythm is SVT with aberrant conduction (Box 10).
- If a second shock (2 J/kg) is unsuccessful or if the tachycardia recurs quickly, consider antiarrhythmic therapy (amiodarone or procainamide) before a third shock (see above) (Box 11).

Tachycardia With Hemodynamic Stability

Because all arrhythmia therapies have the potential for serious adverse effects, consider consulting an expert in pediatric arrhythmias before treating children who are hemodynamically stable.

- For SVT, see above.
- For VT, give an infusion of amiodarone slowly (minutes to an hour depending on the urgency) (Class IIb; LOE 7 [extrapolated from adult studies]) while you monitor the ECG and blood pressure. If there is no effect and there are no signs of toxicity, give additional doses (Table 1). If amiodarone is not available, consider giving procainamide slowly (over 30 to 60 minutes) while you monitor the ECG and blood pressure (Class IIb; LOE 5, 6, 7). Do not administer amiodarone and procainamide together without expert consultation.

Special Resuscitation Situations

Trauma

Some aspects of trauma resuscitation require emphasis because improperly performed resuscitation is a major cause of preventable pediatric death.[174] Common errors in pediatric trauma resuscitation include failure to open and maintain the airway, failure to provide appropriate fluid resuscitation, and failure to recognize and treat internal bleeding. Involve a qualified surgeon early, and if possible, transport a child with multisystem trauma to a trauma center with pediatric expertise.

The following are special aspects of trauma resuscitation:

- When the mechanism of injury is compatible with spinal injury, restrict motion of the cervical spine and avoid traction or movement of the head and neck. Open and maintain the airway with a jaw thrust, and do not tilt the head.

  If you cannot open the airway with a jaw thrust, use head tilt–chin lift, because you must establish a patent airway. Because of the disproportionately large head size in infants and young children, optimal positioning may require recessing the occiput[60] or elevating the torso to avoid undesirable backboard-induced cervical flexion.[59,60]
- Do not overventilate (Class III; LOE 3[175]; 5, 6) even in case of head injury.[176] Intentional brief hyperventilation may be used as a temporizing rescue therapy when you observe signs of impending brain herniation (eg, sudden rise in measured intracranial pressure, dilated pupil[s] not responsive to light, bradycardia, hypertension).
- Suspect thoracic injury in all thoracoabdominal trauma, even in the absence of external injuries. Tension pneumothorax, hemothorax, or pulmonary contusion may impair breathing.
- If the patient has maxillofacial trauma or if you suspect a basilar skull fracture, insert an orogastric rather than a nasogastric tube.[177]
- Treat signs of shock with a bolus of 20 mL/kg of an isotonic crystalloid (eg, normal saline or lactated Ringer's solution) even if blood pressure is normal. Give additional boluses (20 mL/kg) if systemic perfusion fails to improve. If signs of shock persist after administration of 40 to 60 mL/kg of isotonic crystalloid, give 10 to 15 mL/kg of blood. Although type-specific crossmatched blood is preferred, in an emergency use O-negative blood in females and O-positive or O-negative in males. If possible warm the blood before rapid infusion.[178,179]
- Consider intra-abdominal hemorrhage, tension pneumothorax, pericardial tamponade, spinal cord injury in infants and children, and intracranial hemorrhage in infants with signs of shock.[180,181]

Children With Special Healthcare Needs

Children with special healthcare needs[182–184] may require emergency care for their chronic conditions (eg, obstruction of a tracheostomy), failure of support technology (eg, ventilator failure), progression of their underlying disease, or events unrelated to those special needs.[185] For additional information about CPR see Part 11: "Pediatric Basic Life Support."

Ventilation With a Tracheostomy or Stoma

Parents, school nurses, and home healthcare providers should know how to assess patency of the airway, clear the airway, and perform CPR using the artificial airway in a child with a tracheostomy.

Parents and providers should be able to provide ventilation via the tracheostomy tube and verify effectiveness by chest expansion. If you cannot ventilate after suctioning the tube, replace it. If a clean tube is unavailable, perform mouth-to-stoma or mask-to-stoma ventilations. If the upper airway is patent, you may be able to provide effective bag-mask ventilation through the nose and mouth while you or someone else occludes the tracheal stoma.

Toxicologic Emergencies

Overdose with cocaine, narcotics, tricyclic antidepressants, calcium channel blockers, and β-adrenergic blockers poses some unique resuscitation problems in addition to the usual resuscitative measures.

Cocaine

Acute coronary syndrome, manifested by chest pain and cardiac rhythm disturbances (including VT and VF), is the most frequent cocaine-related reason for hospitalization in adults.[186,187] Cocaine prolongs the action potential and QRS duration and impairs myocardial contractility.[188,189]

Treatment

- Cool aggressively; hyperthermia is associated with an increase in toxicity.[190]
- For coronary vasospasm, consider nitroglycerin (Class IIa; LOE 5, 6),[191,192] a benzodiazepine, and phentolamine[193,194] (Class IIb; LOE 5, 6).

- Do not give β-adrenergic blockers.[190]
- For ventricular arrhythmia, consider sodium bicarbonate (1 to 2 mEq/kg)[195,196] (Class IIb; LOE 5, 6, 7) in addition to standard treatments.
- To prevent arrhythmia secondary to myocardial infarction, consider a lidocaine bolus followed by a lidocaine infusion (Class IIb; LOE 5, 6).

Tricyclic Antidepressants and Other Sodium Channel Blockers

Toxic doses cause cardiovascular abnormalities, including intraventricular conduction delays, heart block, bradycardia, prolongation of the QT interval, ventricular arrhythmias (including torsades de pointes, VT, and VF), hypotension,[189,197] seizures, and a depressed level of consciousness.

Treatment

- Give 1 to 2 mEq/kg boluses of sodium bicarbonate until arterial pH is >7.45, and then infuse 150 mEq $NaHCO_3$ per liter of D_5W to maintain alkalosis. In severe intoxication, increase the pH to 7.50 to 7.55.[189,198] Do not administer Class I_A (quinidine, procainamide), Class I_C (flecainide, propafenone), or Class III (amiodarone and sotalol) antiarrhythmics, which may exacerbate cardiac toxicity (Class III; LOE 6, 8).[198]
- For hypotension, give boluses (10 mL/kg each) of normal saline. If you need a vasopressor, epinephrine and norepinephrine have been shown to be more effective than dopamine in raising blood pressure.[199,200]
- Consider extracorporeal membrane oxygenation if high-dose vasopressors do not maintain blood pressure.[201,202]

Calcium Channel Blockers

Manifestations of toxicity include hypotension, ECG changes (prolongation of the QT interval, widening of the QRS, and right bundle branch block), arrhythmias (bradycardia, SVT, VT, torsades de pointes, and VF),[203] and altered mental status.

Treatment

- Treat mild hypotension with small boluses (5 to 10 mL/kg) of normal saline because myocardial depression may limit the amount of fluid the patient can tolerate.
- The effectiveness of calcium administration is variable (Class IIb; LOE 7, 8).[203–207] Try giving 20 mg/kg (0.2 mL/kg) of 10% calcium chloride over 5 to 10 minutes; if there is a beneficial effect, give an infusion of 20 to 50 mg/kg per hour. Monitor ionized calcium concentration to prevent hypercalcemia. It is preferable to administer calcium chloride via a central venous catheter; use caution when infusing into a peripheral IV because of the risk for sclerosis or infiltration.
- For bradycardia and hypotension, consider a high-dose vasopressor such as norepinephrine or epinephrine (Class IIb; LOE 5).[206]
- There is insufficient data to recommend for or against an infusion of insulin and glucose[208–211] or sodium bicarbonate (Class Indeterminate).

β-Adrenergic Blockers

Toxic doses of β-adrenergic blockers cause bradycardia, heart block, and decreased cardiac contractility, and some (eg, propranolol and sotalol) may also prolong the QRS and the QT intervals.[211–214]

Treatment

- High-dose epinephrine infusion may be effective[214,215] (Class Indeterminate; LOE 5, 6).
- Consider glucagon (Class IIb; LOE 5, 6).[211,214,216,217] In adolescents, infuse 5 to 10 mg of glucagon over several minutes followed by an IV infusion of 1 to 5 mg/h. If you are giving >2 mg of glucagon, reconstitute it in sterile water (<1 mg/mL) rather than the diluent supplied by the manufacturer.[217]
- Consider an infusion of glucose and insulin (Class Indeterminate; LOE 6).[208]
- There is insufficient data to make a recommendation for or against using calcium (Class Indeterminate; LOE 5, 6).[204,218,219] Calcium may be considered if glucagon and catecholamine are ineffective (Class IIb; LOE 5, 6).

Opioids

Narcotics may cause hypoventilation, apnea, bradycardia, and hypotension.

Treatment

- Ventilation is the initial treatment for severe respiratory depression from any cause (Class I).
- Naloxone reverses the respiratory depression of narcotic overdose (Class I; LOE: 1[220]; LOE 2[221]; LOE 3[222]; 5, 6[223,224]), but in persons with long-term addictions or those with cardiovascular disease, naloxone may increase heart rate and blood pressure and cause acute pulmonary edema, cardiac arrhythmias (including asystole), and seizures. Ventilation before administration of naloxone appears to reduce these adverse effects.[225] Intramuscular administration of naloxone may lower the risk.

Postresuscitation Stabilization

The goals of postresuscitation care are to preserve brain function, avoid secondary organ injury, diagnose and treat the cause of illness, and enable the patient to arrive at a pediatric tertiary-care facility in an optimal physiological state. Reassess frequently because cardiorespiratory status may deteriorate.

Respiratory System

Continue supplementary oxygen until you confirm adequate blood oxygenation and oxygen delivery. Monitor by continuous pulse oximetry.

Intubate and mechanically ventilate the patient if there is significant respiratory compromise (tachypnea, respiratory distress with agitation or decreased responsiveness, poor air exchange, cyanosis, hypoxemia). If the patient is already intubated, verify tube position, patency, and security. In the hospital setting, obtain arterial blood gases 10 to 15 minutes after establishing the initial ventilatory settings and make appropriate adjustments. Ideally correlate blood gases with capnographic end-tidal CO_2 concentration to enable noninvasive monitoring of ventilation.

Control pain and discomfort with analgesics (eg, fentanyl or morphine) and sedatives (eg, lorazepam, midazolam). In very agitated patients, neuromuscular blocking agents (eg,

TABLE 2. Medications to Maintain Cardiac Output and for Postresuscitation Stabilization

Medication	Dose Range	Comment
Inamrinone	0.75–1 mg/kg IV/IO over 5 minutes; may repeat × 2; then: 2–20 μg/kg per minute	Inodilator
Dobutamine	2–20 μg/kg per minute IV/IO	Inotrope; vasodilator
Dopamine	2–20 μg/kg per minute IV/IO	Inotrope; chronotrope; renal and splanchnic vasodilator in low doses; pressor in high doses
Epinephrine	0.1–1 μg/kg per minute IV/IO	Inotrope; chronotrope; vasodilator in low doses; pressor in higher doses
Milrinone	50–75 μg/kg IV/IO over 10–60 min then 0.5–0.75 μg/kg per minute	Inodilator
Norepinephrine	0.1–2 μg/kg per minute	Inotrope; vasopressor
Sodium nitroprusside	1–8 μg/kg per minute	Vasodilator; prepare only in D_5W

IV indicates intravenous; and IO, intraosseous.
Alternative formula for calculating an infusion:
Infusion rate (mL/h) = [weight (kg) × dose (μg/kg/min) × 60 (min/h)]/concentration μg/mL).

vecuronium or pancuronium) with analgesia or sedation, or both, may improve ventilation and minimize the risk of tube displacement. Neuromuscular blockers, however, will mask seizures.

Monitor exhaled CO_2, especially during transport and diagnostic procedures.[226] Insert a gastric tube to relieve and help prevent gastric inflation.

Cardiovascular System

Continuously monitor heart rate, blood pressure (by direct arterial line if possible), and oxygen saturation. Repeat clinical evaluations at least every 5 minutes until the patient is stable. Monitor urine output with an indwelling catheter.

Remove the IO access after you have alternate (preferably 2) secure venous lines. As a minimum, perform the following laboratory tests: central venous or arterial blood gas analysis and measurement of serum electrolytes, glucose, and calcium levels. A chest x-ray may help you evaluate endotracheal tube position, heart size, and pulmonary status.

Drugs Used to Maintain Cardiac Output (Table 2)

Myocardial dysfunction is common after cardiac arrest.[227,228] Systemic and pulmonary vascular resistance are increased except in some cases of septic shock.[229] Vasoactive agents may improve hemodynamics, but each drug and dose must be tailored to the patient (Class IIa; LOE 5, 6, 7) because clinical response is variable. Infuse all vasoactive drugs into a secure IV line. The potential adverse effects of catecholamines include local ischemia and ulceration, tachycardia, atrial and ventricular tachyarrhythmias, hypertension, and metabolic changes (hyperglycemia, increased lactate concentration,[230] and hypokalemia).

Epinephrine

Low-dose infusions (<0.3 μg/kg per minute) generally produce β-adrenergic action (potent inotropy and decreased systemic vascular resistance), and higher-dose infusions (>0.3 μg/kg per minute) cause α-adrenergic vasoconstriction.[231] Because there is great interpatient variability,[232,233] titrate the drug to the desired effect. Epinephrine may be preferable to dopamine in patients (especially infants) with marked circulatory instability and decompensated shock.

Dopamine

Titrate dopamine to treat shock that is unresponsive to fluid and when systemic vascular resistance is low (Class IIb; LOE 5, 6, 7).[229,234] Typically a dose of 2 to 20 μg/kg per minute is used. Although low-dose dopamine infusion has been frequently recommended to maintain renal blood flow or improve renal function, more recent data has failed to show a beneficial effect from such therapy. At higher doses (>5 μg/kg per minute), dopamine stimulates cardiac β-adrenergic receptors, but this effect may be reduced in infants and in chronic congestive heart failure.[231] Infusion rates >20 μg/kg per minute may result in excessive vasoconstriction.[231]

Dobutamine Hydrochloride

Dobutamine has a selective effect on β_1- and β_2-adrenergic receptors; it increases myocardial contractility and usually decreases peripheral vascular resistance. Titrate an infusion[232,235,236] to improve cardiac output and blood pressure, especially due to poor myocardial function.[236]

Norepinephrine

Norepinephrine is a potent inotropic and peripheral vasoconstricting agent. Titrate an infusion to treat shock with low systemic vascular resistance (septic, anaphylactic, spinal, or vasodilatory) unresponsive to fluid.

Sodium Nitroprusside

Sodium nitroprusside increases cardiac output by decreasing vascular resistance (afterload). If hypotension is related to poor myocardial function, consider using a combination of sodium nitroprusside to reduce afterload and an inotrope to improve contractility.

Inodilators

Inodilators (inamrinone and milrinone) augment cardiac output with little effect on myocardial oxygen demand. Use an inodilator for treatment of myocardial dysfunction with increased systemic or pulmonary vascular resistance.[237–239]

Administration of fluids may be required because of the vasodilatory effects.

Inodilators have a long half-life with a long delay in reaching a new steady-state hemodynamic effect after changing the infusion rate (18 hours with inamrinone and 4.5 hours with milrinone). In case of toxicity, if you stop the infusion the adverse effects may persist for several hours.

Neurologic System

One goal of resuscitation is to preserve brain function. Prevent secondary neuronal injury by adhering to the following precautions:

- Do not provide routine hyperventilation. Hyperventilation has no benefit and may impair neurologic outcome, most likely by adversely affecting cardiac output and cerebral perfusion.[175] Intentional brief hyperventilation may be used as temporizing rescue therapy in response to signs of impending cerebral herniation (eg, sudden rise in measured intracranial pressure, dilated pupil[s] not responsive to light, bradycardia, hypertension).
- When patients remain comatose after resuscitation, consider cooling them to a temperature of 32°C to 34°C for 12 to 24 hours because cooling may aid brain recovery (Class IIb). Evidence in support of hypothermia is LOE 7 (extrapolated from LOE 1[240] and LOE 2[241] studies in adults following resuscitation from VF sudden cardiac arrest and 2 LOE 2 neonatal studies[242,243]). The ideal method and duration of cooling and rewarming are not known. Prevent shivering by providing sedation and, if needed, neuromuscular blockade. Closely watch for signs of infection. Other complications of hypothermia include diminished cardiac output, arrhythmia, pancreatitis, coagulopathy, thrombocytopenia, hypophosphatemia, and hypomagnesemia. Neuromuscular blockade can mask seizures.
- Monitor temperature and treat fever aggressively with antipyretics and cooling devices because fever adversely influences recovery from ischemic brain injury (Class IIb; LOE 4, 5, 6).[244–248]
- Treat postischemic seizures aggressively; search for a correctable metabolic cause such as hypoglycemia or electrolyte imbalance.

Renal System

Decreased urine output (<1 mL/kg per hour in infants and children or <30 mL/h in adolescents) may be caused by prerenal conditions (eg, dehydration, inadequate systemic perfusion), renal ischemic damage, or a combination of factors. Avoid nephrotoxic medications and adjust the dose of medications excreted by the kidneys until you have checked renal function.

Interhospital Transport

Ideally postresuscitation care should be provided by a trained team in a pediatric intensive care facility. Contact such a unit as early into the resuscitation attempt as possible and coordinate transportation with the receiving unit.[249] Transport team members should be trained and experienced in the care of critically ill and injured children[37,250] and supervised by a pediatric emergency medicine or pediatric critical care physician. The mode of transport and composition of the team should be established for each system based on the care required by an individual patient.[251] Monitor exhaled CO_2 (qualitative colorimetric detector or capnography) during interhospital or intrahospital transport of intubated patients (Class IIa).

Family Presence During Resuscitation

Most family members would like to be present during resuscitation.[252–257] Parents and care providers of chronically ill children are often knowledgeable about and comfortable with medical equipment and emergency procedures. Family members with no medical background report that being at the side of a loved one and saying goodbye during the final moments of life is comforting[254,258] and helps in their adjustment,[252] and most would participate again.[254] Standardized psychological examinations suggest that, compared with those not present, family members who were present during attempted resuscitation have less anxiety and depression and more constructive grieving behavior.[257] Parents or family members often fail to ask, but healthcare providers should offer the opportunity whenever possible.[256,258,259] If the presence of family members proves detrimental to the resuscitation, they should be gently asked to leave. Members of the resuscitation team must be sensitive to the presence of family members, and one person should be assigned to comfort, answer questions, and discuss the needs of the family.[260]

Termination of Resuscitative Efforts

Unfortunately there are no reliable predictors of outcome during resuscitation to guide when to terminate resuscitative efforts. Witnessed collapse, bystander CPR, and a short time interval from collapse to arrival of professionals improve the chances of a successful resuscitation. In the past, children who underwent prolonged resuscitation and absence of return of spontaneous circulation after 2 doses of epinephrine were considered unlikely to survive,[1,23,261] but intact survival after unusually prolonged in-hospital resuscitation has been documented.[61,122,262–265] Prolonged efforts should be made for infants and children with recurring or refractory VF or VT, drug toxicity, or a primary hypothermic insult. For further discussion on the ethics of resuscitation, see Part 2: "Ethical Issues."

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